

Original Article

Clinical Characteristics and Comorbidities of Pediatric Atopic Dermatitis in an Algerian Cohort: Relevance to Contemporary Therapeutic Management

Auda Ameur^{1,2}, Wiam Saadi³, Charef Latroch², Ahlam Fatmi⁴, Karim Bouziane Nedjadi², Véronique Desvignes^{5,6}

¹Pediatric Dermatologist, University of Oran 1, Oran, Algeria

²Department of Pediatrics B, Amilcar Cabral, Oran University Hospital, Oran, Algeria

³Department of Biology, Faculty of Nature, Life and Earth Sciences, University of Djillali Bounaama, Khemis Miliana, Algeria

⁴Department of Microbiology & Biochemistry, Faculty of Science, University of M'sila, M'sila, Algeria

⁵Department of Pediatrics, Clermont-Ferrand University Hospital, Clermont-Ferrand, France

⁶Scientific Committee, French Association of Ambulatory Pediatrics (AFPA), Saint-Géréon, France

Received: May 06, 2026

Accepted: Aug 02, 2026

Corresponding author's email:

audaameur@gmail.com

Citation: Ameur A, Saadi W, Latroch C, Fatmi A, Bouziane Nedjadi K, Desvignes V. Clinical Characteristics and Comorbidities of Pediatric Atopic Dermatitis in an Algerian Cohort: Relevance to Contemporary Therapeutic Management. *Epidemiology and Health Data Insights*. 2026;2(5):ehdi051.

<https://doi.org/10.63946/ehdi/19085>

Copyright: By the author(s).

License: A non-exclusive license by the publisher. Published by Australasia Publishing Group LLP.

Open Access: This article is an open access article distributed under the terms and conditions of the CC-BY Creative Commons Attribution license

<https://creativecommons.org/licenses/by/4.0>



ABSTRACT

Background: Atopic dermatitis (AD) is a chronic relapsing inflammatory skin disease characterized by intense pruritus, xerosis, recurrent eczematous lesions, and impaired quality of life. Although AD is common in North Africa, data on its associated comorbidities in Algerian children remain scarce.

Objective: To characterize the clinical profile and associated comorbidities of pediatric atopic dermatitis in an Algerian cohort and to review current therapeutic advances.

Methods: We conducted a retrospective cross-sectional study including 53 children diagnosed with atopic dermatitis according to the Hanifin and Rajka criteria at the Pediatric Dermatology Unit of the University Hospital of Oran, Algeria, between January 2025 and January 2026. Demographic characteristics, clinical manifestations, allergic comorbidities, family history, and quality of life assessed using the Children's Dermatology Life Quality Index (CDLQI) were analyzed.

Results: Atopic dermatitis represented 4.42% of all dermatology consultations during the study period. The mean age was 6.7 years, with a female predominance. Familial atopy was the most frequent comorbidity (90.6%), followed by familial asthma (49.1%) and familial eczema (43.4%). Among personal allergic comorbidities, asthma affected 26.4%, allergic rhinitis 24.5%, and allergic conjunctivitis 11.3% of patients. Personal atopy was significantly associated with asthma (OR = 7.9, 95% CI: 2.46–25.46), food allergy (OR = 6.2, 95% CI: 2.30–17.10), allergic conjunctivitis (OR = 4.1, 95% CI: 1.50–11.29), and allergic rhinitis (OR = 3.5, 95% CI: 1.38–9.22). Quality of life was markedly affected, with sleep disturbances reported in 92.5% of patients, stress and anxiety in 50.9%, reduced daily activities in 34.0%, and school absenteeism in 32.1%.

Conclusion: Pediatric atopic dermatitis in this Algerian cohort was characterized by a high burden of allergic comorbidities, a strong familial atopic background, and a substantial impact on quality of life. Early recognition of associated conditions and timely multidisciplinary management are essential to improve patient outcomes and may help reduce progression along the atopic march.

Keywords: Algeria; Atopic Dermatitis; Skin Immunology; Type 2 Inflammation

Introduction

Atopic dermatitis (AD) is a heterogeneous chronic inflammatory skin disease characterized by recurrent episodes of intense pruritus. It comprises distinct clinical phenotypes and immunological endotypes that may influence disease severity, therapeutic response, and long-term outcomes. AD significantly impairs the quality of life of affected children and their families [1]

Globally, atopic dermatitis represents a major public health concern. In Europe, it affects approximately 10–15% of children and 4% of adults. In Africa, its prevalence ranges from 4.7% to 23%. In Algeria, in 2020, the prevalence was estimated at 5.6% among children under 15 years of age, with a female predominance and a bimodal distribution showing peaks in early childhood and middle adulthood [2].

Recent advances have considerably improved our understanding of the molecular mechanisms underlying atopic dermatitis, highlighting the central role of epidermal barrier dysfunction and type 2 immune inflammation in disease pathogenesis. These mechanisms involve alterations in epidermal barrier integrity, including tight junction defects, together with immune dysregulation, which contribute to disease initiation and persistence [3, 5, 6]. Consequently, the management of AD requires frequent medical follow-up and strict adherence to treatment. Furthermore, childhood AD is often the first manifestation of the so-called “atopic march,” characterized by the sequential

development of allergic diseases such as asthma, allergic rhinitis, and food allergies later in life [4,7].

Several health conditions can coexist with atopic dermatitis, with some manifesting in childhood and others developing later in adulthood, especially among individuals with long-standing disease. Although substantial progress has been made in understanding the epidemiology and pathophysiology of atopic dermatitis worldwide, data from Algeria remain limited, particularly regarding the spectrum of associated comorbidities in pediatric patients. Most published studies have focused on prevalence estimates, whereas information on clinical characteristics, allergic comorbidities, and disease burden among Algerian children is scarce. Given the potential influence of genetic, environmental, and socioeconomic factors on disease expression, locally generated data are essential to improve understanding of atopic dermatitis and to guide patient management. Furthermore, data from Western Algeria are particularly limited. To the best of our knowledge, no previous study has comprehensively evaluated the clinical characteristics and associated comorbidities of pediatric atopic dermatitis in Western Algeria. Therefore, this study aimed to characterize the spectrum of comorbidities associated with atopic dermatitis among patients followed in the pediatric dermatology unit of the University Hospital of Oran.

Materials and Methods

A monocentric cross-sectional analytical study was conducted in the Department of Pediatrics of the University Hospital of Oran between January 2025 and January 2026, following approval by the local ethics committee. All children attending the pediatric dermatology outpatient clinic during the study period and diagnosed with atopic dermatitis according to the Hanifin and Rajka criteria were screened for eligibility. The diagnosis was established by the pediatric dermatologist responsible for the unit, together with the pediatric dermatology team, using the Hanifin and Rajka diagnostic criteria.

Both newly diagnosed and previously known cases were eligible for inclusion, whereas patients with incomplete medical records or those lost to follow-up were excluded. Clinical and demographic data were retrospectively collected from patients' medical records. Comorbidities were considered present when documented by the treating physician. Disease severity was not systematically assessed using validated scoring systems such as SCORAD or EASI.

Quality-of-life data were collected using the Children's Dermatology Life Quality Index (CDLQI), a validated questionnaire designed to assess the impact of skin diseases on children's daily activities, school performance, sleep, leisure activities, personal relationships, and emotional well-being.

The variables analyzed included age, sex, region of origin, disease duration (calculated from symptom onset to the consultation date), reason for consultation, associated comorbidities, family history of atopy, triggering factors for disease exacerbation, and the type and distribution of cutaneous lesions.

Data processing and statistical analyses were performed using SPSS software. Qualitative variables were summarized as frequencies and percentages. Associations between categorical variables were assessed using Pearson's Chi-square test when expected cell counts were adequate, whereas Fisher's exact test was used when expected frequencies were less than five. To evaluate the association between personal atopy and allergic comorbidities, 2×2 contingency tables were constructed. Odds ratios (ORs)

and their corresponding 95% confidence intervals (95% CIs) were calculated from these contingency tables to estimate the strength of associations between personal atopy and the presence of asthma, allergic rhinitis, allergic conjunctivitis, and food allergy. Given the

limited sample size, no multivariable adjustment for potential confounding factors was performed. Statistical significance was defined as a two-sided p-value < 0.05.

Results

During the study period, 78 cases of atopic dermatitis were identified, of which 53 met the inclusion criteria, representing 4.42% of all

dermatology consultations during the study period (Table 1).

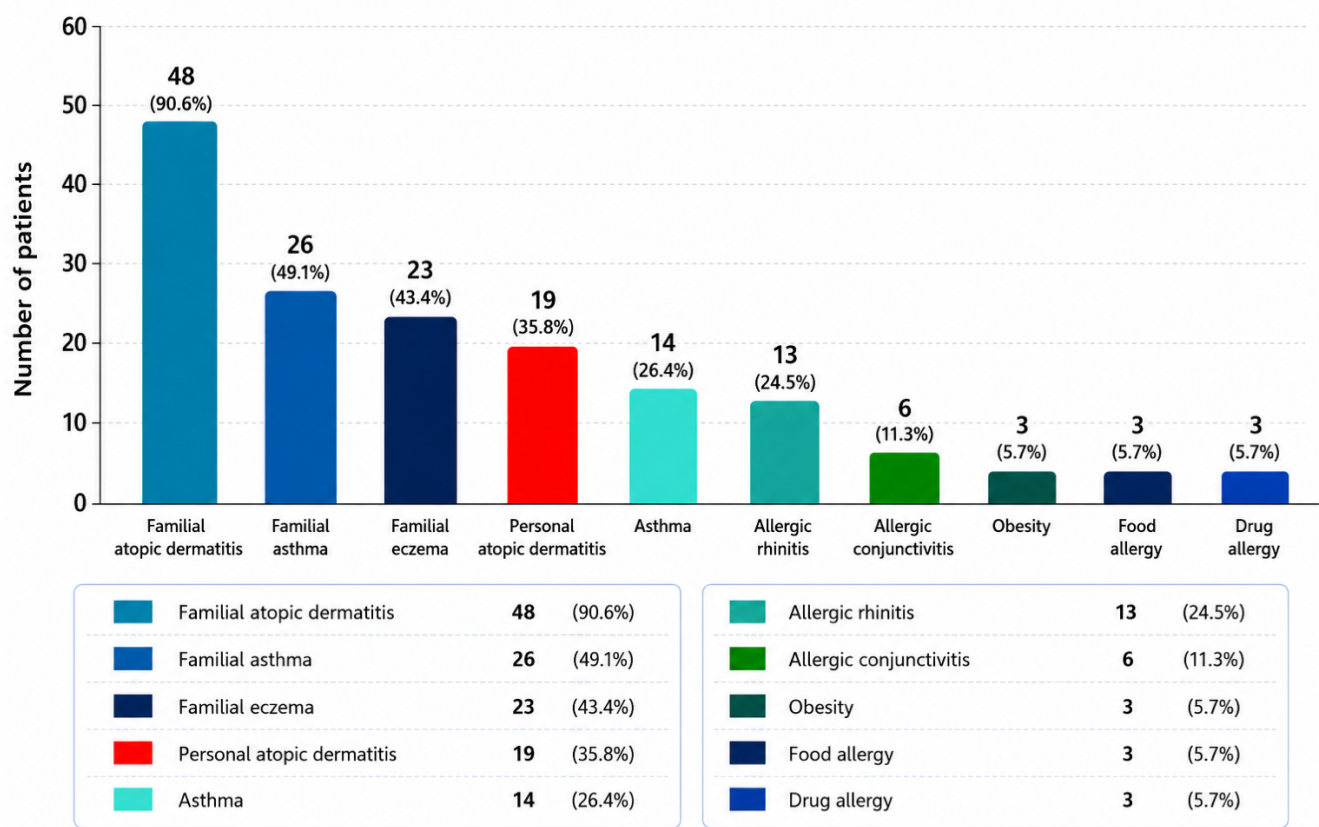
Table 1. Prevalence of atopic dermatitis among dermatology consultations during the study period.

Variable	Number (n)	Percentage (%)
Total dermatology consultations	1200	100
Patients with atopic dermatitis	53	4.42
Other dermatological conditions	1147	95.58

A female predominance was observed, with a sex ratio of 0.87. The mean age of the patients was 6.7 years. The 0–5-year age group was the most represented, accounting for 61.7% of cases, indicating a predominance of younger children in the study population.

The main reason for consultation was skin eruptions, reported in 52 patients (98.11%), frequently associated with pruritus in 49 patients (92.45%). The mean disease duration was 2.8 years.

Regarding comorbidities (Figure 1), 10 types of comorbidities were identified among the 53 patients. A high frequency of familial atopy was observed in 48 patients (90.57%). Familial asthma and familial eczema were reported in 26 patients (49.05%) and 23 patients (43.40%), respectively. Passive smoking exposure was documented in 6 of the 53 patients (11.32%).



Percentages are calculated on the total number of patients (n = 53).

Figure 1. Personal Atopic Comorbidities and Familial Atopic History in Children with Atopic Dermatitis.

Among personal atopic comorbidities (Table 2), asthma was the most frequently reported condition, affecting 14 patients (26.41%), followed by allergic rhinitis in 13 patients (24.52%) and allergic conjunctivitis in 6 patients (11.32%). Significant associations were observed between personal atopy and several allergic comorbidities, including asthma

(OR = 7.9; 95% CI: 2.46–25.46), food allergy (OR = 6.2; 95% CI: 2.30–17.10), allergic conjunctivitis (OR = 4.1; 95% CI: 1.50–11.29), and allergic rhinitis (OR = 3.5; 95% CI: 1.38–9.22). Furthermore, the presence of personal atopy was significantly associated with exacerbations of atopic dermatitis ($p < 0.05$).

Table 2. Association between personal atopy and allergic comorbidities in patients with atopic dermatitis.

Variable	Odds Ratio (OR)	95% Confidence Interval	p-value
Asthma	7.9	2.46–25.46	<0.05
Allergic conjunctivitis	4.1	1.50–11.29	0.003
Allergic rhinitis	3.5	1.38–9.22	0.004
Food allergy	6.2	2.30–17.10	<0.05

Regarding cutaneous manifestations (Table 3), xerosis was the most common dermatological finding, observed in 34 patients (64.15%). Other skin manifestations included erythematous-vesicular lesions

in 13 patients (24.52%), weeping lesions in 7 patients (13.20%), local superinfections in 13 patients (24.52%), lichenified lesions in 9 patients (16.98%), and post-inflammatory hyperpigmentation in 3 patients (5.66%).

Table 3. Frequency of cutaneous manifestations observed in patients with atopic dermatitis.

Cutaneous manifestation	Number of patients	Percentage (%)
Cutaneous xerosis	34	64.15
Erythematous-vesicular lesions	13	24.52
Local superinfections	13	24.52
Lichenified lesions	9	16.98
Weeping lesions	7	13.20
Post-inflammatory hyperpigmentation	3	5.66

Concerning the anatomical distribution of lesions (Table 4), the most frequently affected areas were the limbs (90.57%), followed by the head and neck

region (73.58%) and the face (71.69%). External genital involvement was observed in 12 patients (22.64%). The trunk was also affected in 12 patients (22.64%).

Table 4. Anatomical distribution of lesions in patients with atopic dermatitis.

Location of lesions	Number of patients	Percentage (%)
Limbs	48	90.57
External genitals	12	22.64
Head and neck	39	73.58
Face	38	71.69
Trunk	12	22.64

The disease had a substantial impact on patients' quality of life and daily functioning (Table 5). Sleep disorders were reported in 49 patients (92.45%), stress and anxiety in 27 patients (50.94%), reduced daily activities in 18 patients (33.96%), and school absenteeism in 17 patients (32.08%). The mean CDLQI score was 11.8 ± 5.4 , with a median of 12 (IQR: 8–16),

indicating a moderate-to-severe impairment in quality of life.

Table 5. Impact of atopic dermatitis on quality of life assessed by the Children's Dermatology Life Quality Index (CDLQI).

Variable	Number of patients (n)	Percentage (%)
Sleep disorders	49	92.45
Stress and anxiety	27	50.94
Reduced daily activities	18	33.96
School absenteeism	17	32.08
CDLQI impact category		
No effect (0–1)	2	3.77
Small effect (2–6)	8	15.09
Moderate effect (7–12)	20	37.74
Very large effect (13–18)	15	28.30
Extremely large effect (19–30)	8	15.09

Discussion

Our study sought to characterize the range of comorbidities linked to atopic dermatitis (AD) and to determine the conditions most commonly associated with this disorder among patients in Oran, Algeria. Atopic dermatitis accounted for 4.42% of dermatology consultations in our cohort. This value is comparable to findings from international epidemiological studies. For instance, a multicenter study conducted by Barbarot et al. reported prevalence rates of 2.2% in Germany, 3.5% in Canada, and 4.9% in the United States . These variations may be explained by differences in environmental exposure, genetic susceptibility, lifestyle factors, and diagnostic criteria across populations. [8]

Atopic dermatitis is frequently associated with other allergic diseases, particularly in patients with early-onset disease that persists into adulthood. This clinical progression is supported by the complex interplay between genetic susceptibility, epidermal barrier dysfunction, and immune dysregulation, which promotes chronic skin inflammation and facilitates the development of allergic comorbidities. [6] In our study, several allergic comorbidities were identified, including allergic rhinitis and food allergy. Our findings are consistent with previous reports identifying allergic rhinitis as one of the most common atopic comorbidities in patients with AD and support the concept of the atopic march, in which atopic dermatitis represents the initial manifestation of a progressive allergic disease spectrum that may subsequently include asthma, allergic rhinitis, and food allergies. [4,7]

Environmental factors also play a significant role in the development and exacerbation of AD. According to the literature, both active and passive smoking are associated with an increased prevalence of atopic dermatitis in adults and children . Exposure to

tobacco smoke may alter immune responses and impair skin barrier function, thereby increasing susceptibility to inflammatory skin diseases . In our cohort, passive smoking exposure was documented in six patients (11.32%). According to the literature, both active and passive smoking are associated with an increased prevalence of atopic dermatitis in adults and children [9]. Environmental conditions such as humidity and temperature may also influence skin barrier function and contribute to disease exacerbation [10].

Food allergy was another comorbidity observed in our cohort. Although food allergy has been reported as a frequent comorbidity in children with atopic dermatitis, the relationship between these conditions remains complex. In our study, the specific food allergens were not identified, and no analysis of their association with disease exacerbations was performed. Previous studies suggest that food allergy may contribute to AD symptoms in a subset of patients by triggering immediate hypersensitivity reactions or delayed inflammatory responses following food ingestion. These mechanisms may exacerbate pruritus and scratching, further impairing skin barrier function and perpetuating cutaneous inflammation.

In contrast, the association between AD and cardiovascular disorders remains controversial. Several studies have found no significant relationship between AD and cardiovascular disease . However, Shalom et al. reported a correlation between AD severity and cardiovascular risk factors in an Israeli population . Obesity has been reported as a potential comorbidity of atopic dermatitis. Previous studies have demonstrated an association between increased body mass index and AD, possibly through mechanisms involving chronic low-grade inflammation and immune dysregulation. Similar findings were reported in an American study demonstrating an association between increased body

mass index (BMI) and AD. Zhang et al. also found that overweight and obese individuals had a higher risk of developing AD, possibly due to altered immune regulation and chronic low-grade inflammation associated with obesity [11-13].

The association between diabetes and atopic dermatitis remains controversial. While most studies have not demonstrated a clear relationship, some authors have reported an increased risk of AD among patients with type 1 diabetes. Although most studies report no clear association between AD and type 2 diabetes, a Taiwanese study conducted by Lin in 2016 demonstrated an increased risk of AD among individuals with type 1 diabetes. The relationship between metabolic disorders and AD may involve immune dysregulation and chronic inflammatory pathways shared by both conditions [14].

Neurological comorbidities, including epilepsy, have been reported in the literature among patients with atopic dermatitis. Chen et al. described an increased risk of epilepsy in individuals with AD, although the underlying mechanisms remain

incompletely understood. Chen et al. reported similar findings in a Taiwanese cohort, where patients with AD showed a higher risk of developing epilepsy later in life. The exact mechanisms underlying this association remain unclear; however, both AD and epilepsy have been linked to chronic inflammatory processes and immune dysregulation [15,16].

Beyond comorbidities, atopic dermatitis represents a complex chronic disease characterized by a multifactorial pathophysiology involving genetic predisposition, epidermal barrier dysfunction, immune dysregulation, and environmental triggers. The disease can be classified according to age of onset. (Figure 2) Pediatric AD is typically categorized into very early onset, early onset, and childhood onset forms. The “very early onset” group includes patients who develop symptoms within the first months of life, with many experiencing remission before the age of two. Early onset occurs between two and six years of age, while childhood onset is observed between six and fourteen years.

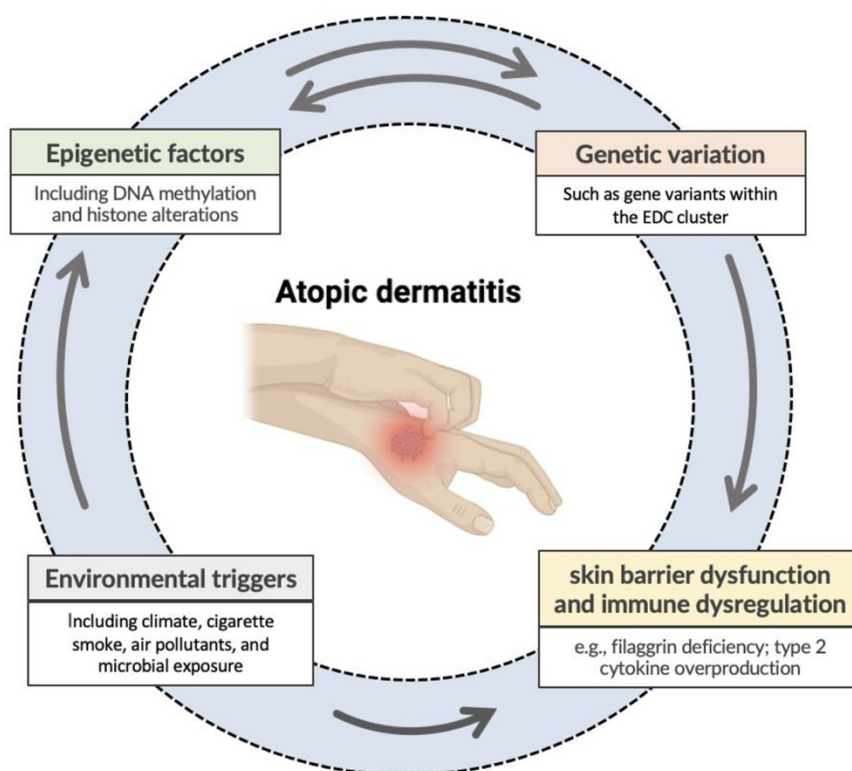


Figure 2. Interacting factors in the pathogenesis of atopic dermatitis. Atopic dermatitis emerges from dynamic interactions between genetic predisposition, epigenetic modifications, and environmental exposures. These interconnected factors contribute to skin barrier dysfunction (e.g., filaggrin deficiency) and immune dysregulation (e.g., Th2-skewed response), which together drive the onset and persistence of the disease.

In the present study, the diagnosis of atopic dermatitis (AD) was established using the Hanifin and Rajka criteria. Although the diagnosis of AD remains primarily clinical, the United Kingdom Working Party

(UKWP) criteria, which represent a simplified and validated adaptation of the Hanifin and Rajka criteria, are widely used in epidemiological studies because of their practicality while maintaining good diagnostic

accuracy. The UKWP criteria require the presence of pruritus associated with at least three additional features, including flexural dermatitis, a history of generalized dry skin, a personal or family history of atopy, and disease onset before the age of two years. However, these criteria may be less applicable in infants, in whom lesions more commonly involve the face, scalp, and extensor surfaces of the limbs. [17]

Pruritus remains the hallmark symptom of AD and plays a central role in the disease burden. Persistent itching triggers the itch-scratch cycle, leading to further disruption of the skin barrier, increased inflammation, and a higher risk of secondary infections. Moreover,

pruritus frequently causes sleep disturbances, anxiety, and psychological stress, significantly affecting patients' quality of life. Studies have demonstrated increased nerve fiber density and hyperactivity in atopic skin, which may contribute to the abnormal sensory perception and chronic itch experienced by patients with AD.

Disease severity is commonly evaluated using validated scoring systems such as the Eczema Area and Severity Index: Eczema Area and Severity Index (EASI) and the SCORing Atopic Dermatitis (SCORAD). These tools allow clinicians to objectively assess disease severity and monitor treatment response (Figure3) [18].



Figure 3. Interacting Clinical Severity of Atopic Dermatitis Across Age Groups. This figure illustrates the varying severity of atopic dermatitis across different age groups. The images depict mild atopic dermatitis in infants (left top), moderate atopic dermatitis in children (right top), and severe atopic dermatitis in adolescents (bottom). Each image highlights the characteristic skin manifestations, including redness, scaling, and thickening, which intensify with severity.

Recent research has also shown that the clinical and immunological features of AD may vary across ethnic groups. For example, Asian patients tend to exhibit stronger Th17 immune polarization, whereas filaggrin gene mutations are more frequently observed

in Caucasian populations and are less common in African populations.

The management of AD requires a comprehensive approach combining non-

pharmacological strategies, topical treatments, and systemic therapies in more severe cases.

Non-pharmacological measures play a crucial role in long-term disease control. Gentle skincare routines, including the use of fragrance-free moisturizers and mild cleansers, are essential to maintain skin hydration and prevent flare-ups. Patients should also avoid known triggers such as irritants, allergens, excessive heat, and psychological stress. Lifestyle modifications, including stress management and dietary adjustments when appropriate, may also contribute to better disease control.

Topical emollients remain the cornerstone of maintenance therapy. These products help restore the epidermal barrier and reduce transepidermal water loss through a combination of emollients, occlusive agents, and humectants. Several clinical trials have demonstrated that regular use of moisturizers reduces pruritus, erythema, fissuring, and lichenification in patients with AD. Early emollient use in neonates has even been associated with a reduction in the cumulative incidence of AD [19,20].

During acute inflammatory flares, topical corticosteroids remain the first-line anti-inflammatory therapy. These medications reduce inflammation by inhibiting cytokine production and antigen presentation. Treatment should be individualized according to patient age, lesion location, and disease severity. Low-potency corticosteroids are preferred for sensitive areas such as the face and skin folds, whereas more potent agents may be used on thicker skin areas for limited periods.

Topical calcineurin inhibitors, including tacrolimus and pimecrolimus, represent an effective steroid-sparing alternative, particularly for sensitive areas. These agents inhibit T-cell activation and reduce the production of inflammatory cytokines. Although concerns have been raised regarding potential long-term malignancy risks, current evidence suggests that this risk remains largely theoretical [21].

For patients with moderate-to-severe AD who do not respond adequately to topical therapy, systemic treatments may be required. These include immunosuppressive agents such as cyclosporine, azathioprine, methotrexate, and mycophenolate mofetil. Phototherapy, particularly narrowband Ultraviolet B (UVB), is another therapeutic option that has demonstrated efficacy in both adults and children.

Conclusion

Atopic dermatitis remains a frequent chronic inflammatory condition associated with considerable clinical and psychosocial consequences in pediatric populations. Our findings demonstrate a high

In recent years, major advances have been achieved with the development of targeted biological therapies. Dupilumab, a monoclonal antibody targeting Interleukin IL-4 and IL-13 signaling pathways, has significantly improved the management of moderate-to-severe AD. By inhibiting the Th2 inflammatory pathway, dupilumab reduces disease severity, pruritus, and flare frequency. This therapy is now approved for use in children, adolescents, and adults with moderate-to-severe disease who require systemic treatment [22].

In Algeria, international recommendations increasingly guide the management of pediatric AD. The recent availability of dupilumab represents an important therapeutic advance for patients with severe disease who are inadequately controlled with conventional treatments.

Despite these therapeutic advances, many patients still experience inadequate symptom control and reduced quality of life. Future research should focus on the identification of biomarkers and personalized treatment strategies to optimize therapeutic outcomes in patients with AD [23,24].

Finally, emerging therapeutic approaches, including Janus kinase (JAK) inhibitors, phosphodiesterase-4 inhibitors, and novel biologics targeting immune pathways such as OX40, may further transform the treatment landscape of AD in the coming years. Advances in nanotechnology-based drug delivery systems may also improve the efficacy and safety of topical treatments.

Limitations

This study has several limitations. The relatively small sample size and single-center design may limit the precision and generalizability of the findings. In addition, the retrospective collection of some clinical data from medical records may have resulted in incomplete information for certain variables. No multivariable analysis was performed; therefore, the reported associations should be interpreted with caution. Disease severity was not systematically assessed using validated instruments such as EASI or SCORAD. However, quality of life was evaluated using the Children's Dermatology Life Quality Index (CDLQI), a validated questionnaire specifically designed for pediatric dermatological conditions. Larger prospective multicenter studies are needed to confirm these findings.

prevalence of allergic comorbidities and a strong familial atopic background among Algerian children with atopic dermatitis.

Early recognition and adequate therapeutic management are essential to improve quality of life and reduce progression toward other atopic disorders. Improved therapeutic education and early

multidisciplinary management may contribute to better disease control and quality of life in children with atopic dermatitis.

Acknowledgements

Author Contributions: Conceptualization: Auda Ameur. Study design: Auda Ameur and Charef Latroch. Data collection: Auda Ameur and Wiam Saadi. Data analysis and interpretation: Auda Ameur and Ahlam Fatmi. Manuscript drafting: Auda Ameur. Critical revision of the manuscript: Charef Latroch, Karim Bouziane Nedjadi, and Véronique Desvignes. All authors reviewed and approved the final version of the manuscript.

Funding: The authors received no specific funding for this work.

Disclosures: The authors declare no competing interests.

Disclosure of AI Use: No artificial intelligence tools were used in the preparation of this manuscript.

References

1. Tokura Y, Hayano S. Subtypes of atopic dermatitis: from phenotype to endotype. *Allergol Int*. 2022;71(1):14–24. <https://doi.org/10.1016/j.alit.2021.07.003>
2. Baghou S, Bensaad D, Taieb A, Ammar-Khodja A. Prévalence et profil clinique de la dermatite atopique en Algérie. *Ann Dermatol Venereol*. 2012;139(12 Suppl):B140. <https://doi.org/10.1016/j.annder.2012.10.200>
3. Baidya A, Mabalirajan U. Pathogenesis and management of atopic dermatitis: insights into epidermal barrier dysfunction and immune mechanisms. *Explor Asthma Allergy*. 2025;3:100973. <https://doi.org/10.37349/ea.2025.100973>
4. Peng W, Novak N. Pathogenesis of atopic dermatitis. *Clin Exp Allergy*. 2015;45(3):566–574. <https://doi.org/10.1111/cea.12495>
5. De Benedetto A, Rafaels NM, McGirt LY, Ivanov AI, Georas SN, Cheadle C, et al. Tight junction defects in patients with atopic dermatitis. *J Allergy Clin Immunol*. 2011;127(3):773–786.e7. <https://doi.org/10.1016/j.jaci.2010.10.018>
6. Sroka-Tomaszewska J, Trzeciak M. Molecular Mechanisms of Atopic Dermatitis Pathogenesis. *Int J Mol Sci*. 2021;22(8):4130. <https://doi.org/10.3390/ijms22084130>
7. Furue M, Ulzii D, Vu Y, Tsuji G, Kido-Nakahara M, Nakahara T. Pathogenesis of Atopic Dermatitis: Current Paradigm. *Iran J Immunol*. 2019;16(2). https://iji.sums.ac.ir/article_40700.html (no DOI available)
8. Barbarot S, Auziere S, Gadkari A, Girolomoni G, Puig L, Simpson EL, et al. Epidemiology of atopic dermatitis in adults: Results from an international survey. *Allergy*. 2018;73(6):1284–1293. <https://doi.org/10.1111/all.13401>
9. Kantor R, Kim A, Thyssen JP, Silverberg JL. Association of atopic dermatitis with smoking: A systematic review and meta-analysis. *J Am Acad Dermatol*. 2016;75(6):1119–1125.e1. <https://doi.org/10.1016/j.jaad.2016.07.017>
10. Engebretsen KA, Johansen JD, Kezic S, Linneberg A, Thyssen JP. The effect of environmental humidity and temperature on skin barrier function and dermatitis. *J Eur Acad Dermatol Venereol*. 2016;30(2):223–249. <https://doi.org/10.1111/jdv.13301>
11. Shalom G, Dreiherr J, Kridin K, Horev A, Khoury R, Battat E, et al. Atopic dermatitis and the metabolic syndrome: a cross-sectional study of 116,816 patients. *J Eur Acad Dermatol Venereol*. 2019;33(9):1762–1767. <https://doi.org/10.1111/jdv.15642>
12. Drucker AM, Li WQ, Lin L, Cho E, Li T, Camargo CA Jr, et al. Atopic dermatitis (eczema) in US female nurses: lifestyle risk factors and atopic co-morbidities. *Br J*

- Dermatol. 2016;174(6):1395–1397. <https://doi.org/10.1111/bjd.14421>
13. Zhang A, Silverberg JI. Association of atopic dermatitis with being overweight and obese: a systematic review and meta-analysis. *J Am Acad Dermatol.* 2015;72(4):606–616.e4. <https://doi.org/10.1016/j.jaad.2014.12.013>
 14. Lin CH, Wei CC, Lin CL, Lin WC, Kao CH. Childhood type 1 diabetes may increase the risk of atopic dermatitis. *Br J Dermatol.* 2016;174(1):88–94. <https://doi.org/10.1111/bjd.14145>
 15. Chen MH, Wu YH, Su TP, Chen YS, Hsu JW, Huang KL, et al. Risk of epilepsy among patients with atopic dermatitis: a nationwide longitudinal study. *Epilepsia.* 2014;55(8):1307–1312. <https://doi.org/10.1111/epi.12668>
 16. Aronica E, Crino PB. Inflammation in epilepsy: clinical observations. *Epilepsia.* 2011;52(Suppl 3):26–32. <https://doi.org/10.1111/j.1528-1167.2011.03140.x>
 17. Williams HC, Burney PG, Pembroke AC, Hay RJ. The U.K. Working Party's Diagnostic Criteria for Atopic Dermatitis. *Br J Dermatol.* 1994;131:383–396. <https://doi.org/10.1111/j.1365-2133.1994.tb08532.x>
 18. Hanifin JM, Thurston M, Omoto M, Cherill R, Tofte SJ, Graeber M. The Eczema Area and Severity Index (EASI): assessment of reliability in atopic dermatitis. *Exp Dermatol.* 2001;10(1):11–18. <https://doi.org/10.1034/j.1600-0625.2001.100102.x>
 19. Sidbury R, Davis DM, Cohen DE, Cordoro KM, Berger TG, Bergman JN, et al. Guidelines of care for the management of atopic dermatitis. *J Am Acad Dermatol.* 2014;71(1):116–132. <https://doi.org/10.1016/j.jaad.2014.03.023>
 20. Simpson EL, Chalmers JR, Hanifin JM, Thomas KS, Cork MJ, McLean WHI, et al. Emollient enhancement of the skin barrier from birth offers effective atopic dermatitis prevention. *J Allergy Clin Immunol.* 2014;134(4):818–823. <https://doi.org/10.1016/j.jaci.2014.08.005>
 21. Hong CH, Papp KA, Lophaven S, et al. Evidence Review of Topical Calcineurin Inhibitors for the Treatment of Atopic Dermatitis. *J Cutan Med Surg.* 2019;23(5):534–547. <https://doi.org/10.1177/1203475419857669>
 22. Napolitano M, Fabbrocini G, Patrino C. Dupilumab Treatment in Children Aged 6–11 Years With Moderate-to-Severe Atopic Dermatitis. *Pediatr Drugs.* 2022;24:315–322. <https://doi.org/10.1007/s40272-022-00524-3>
 23. Libon F, et al. Biomarkers in Atopic Dermatitis: Current State and Future Perspectives. *Int J Mol Sci.* 2024;25(13):7045. <https://doi.org/10.3390/ijms25137045>
 24. Chiricozzi A, Guttman-Yassky E, et al. Enabling Precision Medicine with Biomarkers of Response in Atopic Dermatitis. *Front Med.* 2025;12:1574697. <https://doi.org/10.3389/fmed.2025.1574697>